Severe neurological symptoms following synthetic cannabinoid intoxication

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ABSTRACT

Synthetic cannabinoids are becoming increasingly popular as substances of abuse. However, in the Netherlands synthetic cannabinoid intoxications are rare. We report a 16-year-old male who became deeply comatose and was admitted to the intensive care unit for invasive mechanical ventilation after abuse of an initially unknown drug. Routine toxicology screening with an immunoassay only detected tetrahydrocannabinol, but additional tests with liquid chromatography mass spectrometry revealed synthetic cannabinoid use. This case underlines the challenging diagnosis of synthetic cannabinoid intoxications and the severe complications they can produce.

KEYWORDS

Synthetic cannabinoids, intoxication, coma, drugs of abuse, LC-MS/MS

INTRODUCTION

Synthetic cannabinoids are becoming increasingly popular as drugs of abuse, mainly in Europe and the United States.1 As a result, patients are presenting to emergency departments with unwanted effects of these drugs. Synthetic cannabinoids are sold in colourful packages under various names, including ‘spice’, ‘K2’, ‘crazy monkey’ and ‘chill out’. Synthetic cannabinoids are a chemically very diverse class of drugs and structurally different from tetrahydrocannabinol in natural cannabis, but all substances were designed to act as agonists of cannabinoid receptors (CB1 and CB2). This explains their psychoactive effects.1 Although intoxications with synthetic cannabinoids are globally on the rise, they are still a very rare phenomenon in the Netherlands.2 This case report describes a patient with very severe neurological symptoms after synthetic cannabinoid intoxication and explains why these intoxications can be very difficult to diagnose.

CASE REPORT

A 16-year-old male with no medical history presented to our emergency department. The patient had lost consciousness after smoking cannabis and subsequently a drug called ‘Bonsai’. On arrival to the emergency department physical examination revealed a Glasgow Coma Score of E1M1V1, dilated pupils, unresponsive to light and absence of some of the other brainstem reflexes.
Patients usually have a positive history of abuse only revealed tetrahydrocannabinol, which was confirmed after discharge by LC-MS/MS. This underlines the diagnostic difficulties that arise because synthetic cannabinoids are not detected by the commonly used kits for screening on drugs of abuse. Fortunately, definite confirmation of synthetic cannabinoids by LC-MS/MS is usually not necessary because their use is clear from the anamnestic and treatment is aspecific and mostly supportive.

After acute synthetic cannabinoid intoxication psychoactive symptoms, such as agitation, restlessness, confusion, anxiety and psychosis, are common. Patients usually display physical signs that also occur with cannabis intoxication, including dilated pupils, red conjunctivae, nausea, slurred speech and sweating. Cardiovascular symptoms, such as hypertension, tachycardia and chest pain, may also be present and rarely synthetic cannabinoid intoxications can lead to myocardial infarction, kidney injury or death. Laboratory tests and electrocardiogram are generally normal and in many cases urine toxicology screens are negative for drugs other than tetrahydrocannabinol, which is often used on the same occasion. Importantly, synthetic cannabinoid use is not ruled out by negative drug screening for tetrahydrocannabinol nor confirmed by a positive tetrahydrocannabinol result.

Treatment of synthetic cannabinoid intoxication is usually supportive and determined by the order and magnitude of symptoms. Agitation and confusion can often be managed with reassurance and avoidance of stimulation, while benzodiazepines can be administered for more serious symptoms such as psychosis. Rarely, prolonged sedation or intubation can be necessary to prevent end-organ damage or rhabdomyolysis. Mild intoxications typically last less than eight hours. However, clinical effects and the duration and degree of toxicity depend on the specific compound used.

In conclusion, synthetic cannabinoid intoxications are on the rise in Europe and the United States. Although they are still very rare in the Netherlands, it is quite possible that Dutch emergency departments will be increasingly confronted with synthetic cannabinoid intoxications. Symptoms can be of greater magnitude and duration compared with cannabis intoxication. This patient presented with severe neurological symptoms including absence of some brain stem reflexes. Mechanical ventilation was necessary. Diagnosis of a synthetic cannabinoid intoxication can be challenging because it is not revealed by most urine drugs of abuse kits. Confirmation is possible with LC-MS/MS, but this technique is costly and is not readily available in most cases.
DISCLOSURES

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